Seizure (2008) **17**, 595—603

ELSEVIER

SEIZURE

www.elsevier.com/locate/yseiz

Psychological treatment of patients with psychogenic non-epileptic seizures: An outcome study

Jarl Kuyk*, **Mieke C. Siffels, Patricia Bakvis, Wilhelmina A.M. Swinkels**

Department of Psychology, Epilepsy Institute of the Netherlands Foundation (SEIN), Heemstede, The Netherlands

Received 7 November 2007; received in revised form 23 January 2008; accepted 29 February 2008

KEYWORDS
Psychogenic non-epileptic seizures;
Treatment outcome;
Psychopathology;
Coping;
Dissociation;
Seizure frequency

Summary

Introduction: It is estimated that up to 25% of patients referred to specialised epilepsy centers suffer from psychogenic non-epileptic seizures (PNES). The prognosis is unfavourable and there are no generally accepted treatment protocols.
Method: In this study, the effect of an uncontrolled, prospective inpatient treatment program for PNES patients is evaluated. The treatment is multidisciplinary and based on cognitive behavioural principles. Seizure control, general psychopathology, anxiety, depression, coping, dissociation and health related quality of life are evaluated. Twenty-two patients participated in the study of which 16 patients were followed 6 months after treatment.
Results: After follow-up, 81% of patients had a seizure reduction of over 50%, and half of them became seizure-free. Measures of anxiety, depression and dissociation tended to normalize, coping was more adequate and health related quality of life was increased slightly. In the period between the end of treatment and follow-up the most positive effects are maintained and even strengthened. Patients who became seizure-free at follow-up improved more on the psychological outcome measures than patients with continuing seizures.
Conclusion: The outcome suggests effectiveness of the treatment. PNES patients may profit from a comprehensive, multidisciplinary treatment program following cognitive behavioural principles. Seizure cessation appears to be an important factor in the improvement of psychological functioning.

* Corresponding author at: PO Box 540, 2130 AM Hoofddorp, The Netherlands. Tel.: +31 235588000; fax: +31 235588279.
E-mail address: jkuyk@sein.nl (J. Kuyk).

1059-1311/$ — see front matter
doi:10.1016/j.seizure.2008.02.006

Introduction

Seizures, i.e. paroxysmal, involuntary disturbances of controlling motor, sensory, autonomic, cognitive, emotional or behavioural functions, may have a serious negative impact on daily life, whether they result from organic or psychological origin. When seizures are caused by excessive and simultaneous electrical discharges of groups of brain cells it is an epileptic event. If there is no epilepsy-characteristic electrical brain activity during the seizure, and no other medical disorders associated with seizure-like events, the seizures are considered as a psychological manifestation.[1] Several authors emphasize the enormous load of psychogenic non-epileptic seizures (PNES) patients within the health service.[2,3] The prevalence of patients with PNES is calculated between 1/3000 and 1/50,000.[4] It is estimated that up to 25% of patients referred to specialised epilepsy centers suffer from PNES.[5] A non-published report from our centre reveals that about 22% of patients admitted for seizure diagnosis do have PNES; another 7% have PNES in combination with epilepsy.

The time between the age at onset of seizures and the correct diagnosis is often several years[6] and many patients are treated with ineffective and potentially toxic anti-epileptic drugs (AEDs), also experiencing the negative psychological and socio-economic consequences of carrying a diagnosis of epilepsy.

There is no evidence that suffering from PNES refers to a unified pathological syndrome or a determined aetiology.[7] Bowman and Markand[8] propose four pathways to the development of PNES, namely a history of childhood physical or sexual abuse, recent sexual assault, multiple life stresses that overwhelmed the patients' coping abilities, and panic attacks mistaken for PNES. This implies that the specificity of any treatment protocol for PNES patients can only be moderate and that treatment programs need to be individualised.[9]

An effective treatment, leading to long-term seizure freedom and a normalized quality of life does not yet exist. Several psychological interventions are described, such as cognitive behavioural therapy, group psycho-education, psychotherapy, eye movement desensitization and a combination of psychological interventions[10–14] (for reviews also see Refs. 7,15,16). In general, the prognosis is unfavourable[15] and seems dependent on factors such as an early diagnosis, psychiatric comorbidity, an acute trauma preceding the onset of seizures, and the duration of seizure history.[9,17,18]

LaFrance et al.[7,15] reviewed PNES treatment studies and concluded that higher success rates in PNES outcome studies were found in longer inpatient admissions and in cases where patients were managed by a multidisciplinary team familiar with PNES. McDade and colleagues[14] show in an uncontrolled inpatient outcome study a cessation of seizures in 50% of patients (n = 16), a decrease of seizure frequency in 19% and no change in 31% of patients after a follow-up of 12 months. Kim et al.[19] evaluated an inpatient treatment program in 14 patients and concluded that 79% experienced either a significant improvement or complete cessation of seizure activity. An inpatient outcome study by Rush et al.[9] showed that patients with a mean duration of seizures of less than 12 months were seizure-free after treatment, while from patients with a longer existent history of seizures, 60% (n = 15) were seizure-free after treatment, suggesting that a longer history of seizures brings about poorer treatment outcome.

In PNES treatment studies, the most frequently reported outcome measure is the change in seizure frequency. One study[20] showed however that seizure cessation rather than seizure reduction results in a better functional outcome. Another study[21] revealed that seizure-free patients may continue to have symptoms of psychopathology, remain unproductive and dependent on social assistance, suggesting that seizure freedom should not be the only focus of treatment.

In this paper, the outcome of an uncontrolled prospective study of the efficacy of an open psychotherapeutic inpatient treatment program is described. The program is individualised and adjusted to the patients' individual needs. The program, consisting of individual and group therapies, focuses on seizure control as well as to the improvement of psychological functioning and psychological well-being.

In this paper, we evaluate seizure frequency, health related quality of live (HRQOL), anxiety, depression, coping and dissociation at the beginning and completion of the treatment, and after a follow-up period of 6 months. To evaluate the relevance of seizure cessation versus seizure reduction, a comparison of these outcome variables will be made between seizure-free patients and patients who were not seizure-free at follow-up. Additionally, the effect of the treatment program will be investigated in each subgroup separately.

Method

Patients and procedure

The treatment program is offered in a special unit of the Epilepsy Institute of The Netherlands

Foundation (SEIN), Heemstede, The Netherlands. PNES patients can be admitted after the diagnosis 'no epilepsy' is made by the neurologist (based on one or more EEG-confirmed typical seizures). Patients are informed that the seizures are not epileptic and cannot be attributed to other medical conditions. They then receive information about the possibilities of treatment. If they agree with admission, the program in the first 4 weeks is mainly focussed on the psychological diagnostic process. Assessment of patients' skills, abilities, psychopathology, and family dynamics is performed to generate a working hypothesis concerning the underlying problems which may cause and maintain the seizures, and to provide the patient with an acceptable explanation of the seizures. Patients are actively involved in this process and this helps them to make the switch from a neurological to a psychological interpretation of their seizures. Following the diagnostic phase, a multidisciplinary treatment is offered based on cognitive behavioural principles. The treatment is aimed at cognitive restructuring, trauma treatment, stimulus differentiation, coping skills, stress management and includes individual and group therapies. Treatment includes individual psychotherapy, psychomotor and creative therapy, family therapy and participation in the following group therapies: assertivity training, rational emotive therapy and training in behavioural analysis. During the weekends the unit is closed and patients go home in order to facilitate the integration of learned skills in their natural environment.

PNES patients who entered this study were admitted to SEIN's psychotherapeutic treatment unit in the period June 2002 to December 2004. Twenty-nine patients agreed to participate, of whom 3 patients stopped treatment prematurely (2 patients because of private reasons and 1 patient disagreed with the treatment plan and left the clinic). This resulted in 26 patients who were included in the study, of which 4 patients suffered from PNES as well as epileptic seizures. These 'mixed-seizure' patients were excluded from the analysis because PNES and epileptic seizures are not always easy to discern from each other. Also, there are indications that the psychiatric profile of this patient group differs from PNES-only patients.[22]

In all patients, the seizure diagnosis was based on EEG/video-registration of one or more typical seizures. At the start of treatment (T1) and at discharge (T2), psychological data were gathered and seizure frequency was observed. The same was done after 6 months (T3). At the time of follow-up, 3 patients were untraceable and 3 patients withdrew from the study.

Measures

Seizure frequency was observed and counted at the time of T1 and T2 by the nursing staff, and at T3 reported by the patients themselves. At T1 and T2 the average seizure frequency per week for the period of the last 3 weeks was registered, and at T3 the mean frequency per week over the last 4 weeks. At T3 self-reported measures of seizure duration and the feeling of seizure control were collected. Also, the number of anti-epileptic medication taken by the patients at T1, T2 and T3 was recorded.

The following questionnaires were used as outcome variables: the Symptom Checklist-90 (SCL-90),[23,24] a multi-dimensional index of psychopathology; the Beck Depression Inventory (BDI), a short self-report questionnaire assessing the degree of depression[25,26]; the State-Trait Inventory (STAI),[27,28] a self-report measure of subjective state and trait anxiety; the Utrecht Coping List (UCL),[29] a Dutch questionnaire measuring coping behaviour; the SF-36,[30,31] measuring self-perceived health related quality of life; the Dissociation Questionnaire (DISQ),[32] measuring self-reported daily dissociative experiences.

Statistical analysis

Data were analyzed using SPSS for Windows release 14. Change in outcome measures was evaluated with the Wilcoxon Matched Pairs Signed Rank test because data were not normally distributed.

Pearson's correlations are calculated to measure the relation between outcome variables and seizure frequency. Fisher's Exact tests are used when comparing seizure-free patients and patients with ongoing seizures.

Results

Patient characteristics

Demographic data are shown in Table 1. In 13 PNES patients the diagnosis PNES was made just before admission. The time between the diagnosis and start of the treatment for the other patients varied from 3.5 to 12 months (mean 6.7 months).

The duration of the program varied from 2 to 6 months with an average of 4.8 months. The mean duration of pre-treatment seizures was 6.7 years (median 4.5 year, range 0—24 years).

Table 1 Patient characteristics

Sex (%)	Female/male	77.3/22.7
Age (years)	Mean (S.D.)	30.6 (10.8)
	Range	19—52
Marital status (%)	Married/living together	40.9
	Single	59.1
Occupation (%)	Housekeeping	9.1
	Employment	54.5
	Disability pension/welfare	27.3
	Other	9.1
Education (%)	Primary education	13.6
	Secondary education	72.8
	Higher education	13.6
Age at onset seizures (years)	Mean (S.D.)	23.9 (11.3)
	Range	8—52
Duration of seizures (years)	Mean (S.D.)	6.7 (7.2)
	Range	0—29

Outcome measures

Seizures

Changes in seizure frequency at T2 and T3 are shown in Table 2. Mean seizure frequency a week at T2 and T3 decreased significantly with respect to the previous measurement. Two patients had no seizures in the period of 3 weeks before T1. At T2 another 4 patients were seizure-free. After 6 months, 7 patients (44%) of the 16 responders reported no seizures during the last 4 weeks, one of whom reported no seizures at T1. In another 6 patients (37.5%) the seizure frequency declined more than 50%. An increase in seizures was experienced by 2 patients.

At follow-up, 6 patients (67%) with ongoing seizures reported less serious seizures than before, 5 (56%) reported that their seizures were of shorter duration, and 7 patients (81%) reported increased control over their seizures.

Anti-epileptic medication

On admission, AEDs were taken by 63.6% of patients (8 patients 1 AED, 6 patients 2 AEDs). After treatment, 2 patients received 1 AED: lamotrigine was prescribed to 1 patient as a precaution because he had paroxysmal EEG abnormalities (which did not correlate with seizure-like manifestations), another patient received valproate for complaints of migraine. At T3 anti-epileptic medication was not changed: 14 patients received no AEDs, 2 patients received 1 AED for reasons mentioned above.

Psychological questionnaires

In Table 3 mean scores are shown in all psychological questionnaires. Patients showed improvements between T1 and T2 on the BDI, STAI-trait, UCL-active and dissociation scores. After follow-up, the improvement seems to increase as the mean scores on all measures decreased. On the SCL-90 and DISQ, significant improvements were found on all subscales. The mean score at T1 on the SCL-90 is relatively high (percentile 97). On T2 the mean scores drop to percentile 87 and on T3 to percentile 69. Mean BDI-scores are located in the 'mild to moderate' severity categories and decrease on T2 and T3 to the minimal depressed category. STAI-anxiety scores are situated in the 8th to 9th decile at T1, in the 7th decile at T2 and in the 4th to 5th decile at T3.

Table 2 Seizure frequency per week at T1 (start of treatment), T2 (end of treatment) and T3 (follow-up)

	T1 (n = 22)	T2 (n = 22)	T3 (n = 16)	z[a] (p) T1 vs. T2	z[a] (p) T2 vs. T3	z[a] (p) T1 vs. T3
Mean (S.D.)	6.6 (9.8)	3.0 (4.7)	0.9 (1.8)	−2.33 (0.02)	−1.99 (0.05)	−3.12 (0.002)
Median	2.5	0.8	0.3			
Range	0—36	0—19	0—11			
No seizures	7.7%	27.3%	44%			
Decrease >50%[b]		40.9%	37.5%			
Decrease <50%[b]		18.2%	6.2%			
Increase[b]		14%	12.5%			

[a] Wilcoxon Matched Pairs Signed Rank, two tailed test.
[b] Change in seizure frequency on T2 and T3 compared with T1.

Table 3 Outcome measures on start of treatment (T1), end of treatment (T2) and follow-up (T3)

	T1 (n = 22) mean (S.D.)	T2 (n = 22) mean (S.D.)	T3 (n = 16) mean (S.D.)	z^a (p) T1 vs. T2	z^a (p) T2 vs. T3	z^a (p) T1 vs. T3
SCL-90	178.7 (57.3)	155.9 (58.5)	135.1 (37.4)	−1.53 (.12)	−1.03 (.30)	−3.00 (.003)
BDI	19.7 (9.4)	11.5 (10.9)	9.2 (7.5)	−3.01 (.003)	−1.08 (.28)	−3.47 (.001)
STAI-state	46.1 (11.9)	40 (11.8)	33.3 (9.1)	−1.63 (.10)	−1.37 (.17)	−3.05 (.002)
STAI-trait	47.2 (10.9)	41.2 (10.9)	36.7 (10.1)	−2.17 (.03)	−0.83 (.41)	−2.33 (.02)
UCL-active	34.6 (8.1)	39 (6.2)	40.3 (5.9)	−2.52 (.01)	−0.31 (.75)	−2.11 (.03)
UCL-passive	63.5 (6.9)	60.3 (10.3)	57.1 (6.9)	−1.01 (.31)	−0.85 (.39)	−2.59 (.01)
DISQ	1.86 (0.37)	1.69 (0.42)	1.48(0.17)	−1.99 (.05)	−0.80 (.42)	−2.59 (.01)

[a] Wilcoxon Matched Pairs Signed Rank, two tailed test.

Quality of life

HRQOL scores on the SF-36 are shown in Table 4. No significant changes are found after treatment at T2. At T3 better scores are obtained in comparison to T1 on the dimension 'role limitation due to emotional problems'.

Seizure frequency, psychopathology and HRQOL

At follow-up, seizure frequency for the whole group showed two significant correlations on the outcome measures: patients with more seizures showed less active coping abilities (r = −0.52; p = 0.039), and had a poorer outcome on the HRQOL dimension 'energy vitality' of the SF-36 (r = −0.56; p = 0.025).

Seven patients had no seizures at T3 and 9 patients reported still having seizures at follow-up. Prior to treatment, both groups did not differ on the patient characteristics and on the outcome measures.

Table 5 shows the changes between T1 and T3 in both groups. Compared to patients with ongoing seizures, seizure-free patients showed improvements on all psychological questionnaires, except for passive coping and dissociation. Although all scores on the HRQOL dimension improved within the seizure-free group compared to T1, no significant differences were found. Patients with continuing seizures improved on general psychopathology, depression and passive coping.

A comparison between seizure-free patients and those not seizure-free on the outcome measures at T3 is shown in Table 6. Seizure-free patients showed significantly less general psychopathology, depression, anxiety and dissociation, and a better active coping than patients with ongoing seizures. Also, the level of the HRQOL dimensions 'mental health', 'energy vitality' and 'pain' were improved on the SF-36, compared to those patients who continued to have seizures.

Discussion

The aim of this study was to evaluate the outcome of an open inpatient treatment program for PNES patients. The overall results suggest that patients profited from the treatment and that positive effects were maintained, and even strengthened, during the follow-up period. The positive effects of treatment were supported by a significant decrease in seizure frequency and decreased scores on mea-

Table 4 Health related quality of life as measured on the SF-36 at start of treatment (T1), end of treatment (T2) and follow-up (T3)

SF36	T1 (n = 19)	T2 (n = 19)	T3 (n = 15)	z^a(p) T1 vs. T2	z^a(p) T2 vs. T3	z^a(p) T1 vs. T3
Physical functioning	78.2	80.8	76.3	−0.84 (.40)	−1.17 (.24)	−0.53 (.59)
Role limitation due to physical problems	47.2	66.7	51.7	−1.74 (.08)	−1.31 (.19)	−0.27 (.78)
Role limitation due to emotional problems	55.6	81.5	84.4	−1.86 (.06)	−0.32 (.75)	−2.21 (.03)
Social functioning	55.6	60.8	68.9	−0.87 (.38)	−0.90 (.92)	−1.68 (.09)
Mental health	62.5	69.3	74.1	−1.60 (.11)	−1.34 (.18)	−1.91 (.06)
Energy vitality	56.8	59.5	54.7	−0.45 (.65)	−1.14 (.25)	−0.60 (.55)
Pain	58.5	68.4	65.2	−1.45 (.15)	−0.77 (.44)	−0.71 (.48)
General health perception	54.9	65.2	60.0	−1.85 (.06)	−1.65 (.10)	−0.94 (.35)
Change in health	53.9	63.2	65.0	−1.22 (.22)	−0.89 (.89)	−1.53 (.13)

[a] Wilcoxon Matched Pairs Signed Rank, two tailed test.

Table 5 Changes in scores on the psychological questionnaires between T1 and T3 in seizure-free and not seizure-free patients

	Seizure-free (n = 7) mean (S.D.)			Not seizure-free (n = 9) mean (S.D.)		
	T1	T3	p[a]	T1	T3	p[a]
SCL-90	176.9 (70.3)	110.1 (11.9)	0.016	185.7 (56.1)	154.4 (39.3)	0.039
BDI	15.3 (9.6)	4.7 (3.1)	0.031	21.7 (10.0)	12.7 (8.1)	0.004
STAI-state	46.0 (12.9)	28.3 (7.8)	0.031	45.3 (12.1)	37.2 (8.4)	0.055
STAI-trait	44.0 (12.8)	30.6 (6.1)	0.031	47.9 (10.1)	41.6 (10.1)	0.36
UCL-active	32.6 (7.6)	42.7 (5.5)	0.031	38.3 (8.4)	38.4 (5.8)	0.69
UCL-passive	63.1 (9.1)	54.9 (8.2)	0.22	64.1 (6.1)	58.8 (5.8)	0.02
DISQ	1.75 (0.15)	1.33 (0.09)	0.12	1.87 (0.40)	1.56 (0.15)	0.08
SF36	Seizure-free (n = 6) mean (S.D.)			Not seizure-free (n = 9) mean (S.D.)		
	T1	T3	p[a]	T1	T3	p[a]
Physical functioning	85.0 (13.4)	90.7 (8.3)	0.37	78.3 (14.4)	65.6 (32.2)	0.28
Role limitation due to physical problem	62.5 (49.4)	75.0 (38.2)	0.75	44.4 (39.1)	38.9 (39.7)	0.93
Role limitation due to emotional problem	77.8 (27.2)	95.2 (12.6)	0.50	48.1 (44.4)	77.8 (33.3)	0.12
Social functioning	48.1 19.4)	79.4 (20.7)	0.06	59.2 (19.2)	62.9 (15.7)	0.70
Mental health	68.7 (21.1)	83.4 (11.2)	0.22	60.4 (17.7)	68.9 (14.9)	0.18
Energy vitality	56.7 (19.7)	77.1 (16.3)	0.09	47.8 (20.2)	41.7 (22.8)	0.40
Pain	61.1 (28.8)	79.4 (28.3)	0.25	60.5 (17.7)	54.3 (22.5)	0.72
General health perception	46.8 (21.5)	69.6 (29.0)	0.22	54.7 (17.6)	53.9 (19.1)	0.73
Change in health	62.5 (20.9)	78.6 (26.7)	0.25	44.4 (16.7)	52.8 (31.7)	0.48

[a] Fisher Exact test.

sures of anxiety, depression, dissociation and an improvement of coping abilities. Six months after discharge, 80% of patients had a seizure reduction of over 50%, and half of them was seizure-free. With regard to HRQOL, PNES patients improved slightly on domains concerned with mental functioning. A decrease in the use of anti-convulsive medication — from 63.6% of patients at admission to 9% at follow-up — may indicate that the treatment brings about a reduction of costs to medical health

Table 6 Outcome at T3 in patients who are seizure-free and those who are not

	Seizure-free (n = 7) mean (S.D.)	Not seizure-free (n = 9) mean (S.D.)	p[a]
SCL-90	110.2 (11.9)	154.4 (39.3)	0.011
BDI	4.7 (3.1)	12.7 (8.1)	0.024
STAI-state	28.3 (7.8)	37.22 (8.4)	0.056
STAI-trait	30.6 (6.1)	41.6 (10.1)	0.024
UCL-active	42.7 (5.5)	38.4 (5.8)	0.048
UCL-passive	54.9 (8.2)	58.8 (5.8)	0.24
DISQ	1.3 (0.09)	1.6 (0.15)	0.044
SF36	Seizure-free (n = 6) mean (S.D.)	Not seizure-free (n = 9) mean (S.D.)	p[a]
Physical functioning	90.7 (8.4)	65.6 (32.1)	0.11
Role limitation due to physical problems	75.0 (38.2)	38.9 39.7)	0.12
Role limitation due to emotional problems	96.2 (12.6)	77.8 (33.3)	0.34
Social functioning	79.4 (20.7)	62.9 (15.7)	0.21
Mental health	83.4 (11.2)	68.9 (14.9)	0.04
Energy vitality	77.1 (16.3)	41.7 (22.8)	0.004
Pain	79.4 (28.3)	54.3 (22.5)	0.05
General health perception	69.6 (29.0)	53.9 (19.1)	0.11
Change in health	78.6 (26.7)	52.8 (31.7)	0.12

[a] Fisher Exact test.

services. General psychopathology, depression and anxiety scores before treatment were increased compared to norms of the healthy population. After treatment and follow-up, the level of general psychopathology was considerably lower, depression was in the minimal range and anxiety was nearly normalized.

Although our results seem in line with other inpatient treatment studies, post-treatment seizure control in our study is difficult to compare with other inpatients treatment studies, due to differences in several patient characteristics. For example, in the Rush study,[9] 65% of patients were seizure-free after a 6 months follow-up, but most seizure-free patients had a PNES history of less than 12 months, which is considered as a favourable prognostic factor. In our study only 1 patient had a seizure history of less than 12 months and the mean seizure duration was 6.7 years. Patients with comorbid epilepsy participated in the studies of Rush[9] and Kim.[19] We excluded them because it was not possible to determine the seizure frequency in a reliable way. McDade[14] also included patients with an IQ $<$ 80 and found a significant association between low IQ and outcome. In our study patients with an IQ $<$ 80 were excluded from treatment at this unit.

Several studies emphasize the importance of seizure cessation rather than a freedom from them.[20,21] Reuber et al. showed that seizure cessation is not a sufficient condition for a good psychosocial outcome in PNES patients, while some of them continue to report symptoms of psychopathology and remain unproductive.[21] In our series, however, psychopathology in seizure-free patients tended to normalize. In patients with continuing seizures, the level of psychopathology improved slightly. The seizure-free group profited more from the treatment than those patients who did not become seizure-free. As psychological well-being seems to be related to seizure cessation, seizure freedom has to be an important goal in the treatment of PNES patients.

Surprisingly, the HRQOL seems to improve only to some extent, despite the improvements in seizure frequency and psychopathology. The exact reason for this result is not quit clear; a replication of our study with more patients may provide more clarity. Considering, however, the long time that most patients were treated as having epilepsy (average of 6.7 years), it can be speculated that the medical diagnosis of epilepsy before correct diagnosis and appropriate treatment of PNES, might have long (psychosocial) after-effects. Therefore, in future studies, it seems of importance to evaluate the effect of treatment on psychosocial factors for a longer time than 6 months.

The first 4 weeks of the program are mainly focused on the psychological diagnostic process. In our opinion, this is an important part of the treatment: patients have the opportunity to gradually get accustomed to the idea that their problem is not somatic but has a psychological origin and, at the same time, experience that their seizures are being taken very seriously. The patients start 'blank' (we only know it is not epilepsy), and together with the patient and family we try to understand and explain the background of the seizures. Our experience with this diagnostic program has been that patients are less inclined to return to a somatic interpretation of the seizures.

This study has several limitations. First, it concerns a small number of patients, which limit solid conclusions. Also, it did not follow a randomized controlled design. We cannot exclude that the inpatient nature of treatment may have resulted in a reduction of stress levels and enhancement of psychological functioning without any further treatment. However, in PNES patients we feel that the most suitable moment to start treatment is when the patient is informed about the seizure diagnosis. It is believed that telling the diagnosis is the first step in treatment[33] and a longer delay between diagnosis and treatment may intensify psychological resistance to the diagnosis and the willingness to be treated. Another problem of randomization is the necessity of a control group, often a waiting list group, or a group treated with 'neutral' interventions. It cannot be excluded that, for example, a delay in treatment can have negative consequences for the course of the seizure disorder or that disappointment in being assigned to a waiting list group is a negative and uncontrolled factor.[34] Another difficulty in performing a randomized design is the heterogeneity of PNES patients: several underlying causes are assumed and the psychiatric comorbidity is variable. For this reason, several authors advocate that treatment programs need to be individualised based on aetiology, level of intelligence, family dynamics, comorbid psychiatric illness and other factors.[9] Possibly, for these reasons controlled treatment effect-studies in PNES patients are scarce and literature about treatment concern mostly case reports or case series (for reviews see Refs. 7,15). A recent Cochrane review by Baker and colleagues[16] found only three controlled randomized studies. These studies however concerned 'conversion' patients, of whom some suffered from PNES.

Our patient group was a selected group, namely, those patients who accepted the inpatient program. Moreover, other patients were not asked or not accepted for treatment. In the study period, 45 patients with the diagnosis PNES were not admitted

to the treatment unit. Eleven of them were referred back to their own doctors and 6 patients were referred to external ambulatory treatment, often because hospitalization was not possible because of private reasons. Severe psychiatric comorbidity was observed in 6 and somatic problems in 3 patients. This was seen as contra-indication for treatment. Two patients had an IQ lower than 80 points and 1 patient could not express herself in the Dutch language. Sixteen patients refused treatment (8 patients disputed the diagnosis; 2 patients stopped admission after the diagnosis and 6 patients wished to seek treatment elsewhere). There is therefore probably a question of inclusion bias with regard to this study. There is some evidence that long-term seizure control depends more on the presence or absence of underlying psychiatric diagnoses than it does on treatment,[35] so it cannot be excluded that relatively 'easy' patients participated in this study.

The duration of this inpatient treatment program is at average 4.8 months and therefore costly. LaFrance et al. note in their reviews that longer inpatient multidisciplinary treatment show higher success rates[7,15] crucial for treatment success is acceptation of the diagnosis and recognition of a psychological explanation for the seizures by the patient and relatives. The changeover from a medical to a psychological attitude to treatment is essential and often difficult for the patient and their environment. In our opinion, this can be at best achieved by 'striking the iron when it is hot' by starting an extensive inpatient treatment immediately after PNES is diagnosed. It is conceivable that, if these conditions are met, outpatient continuation of treatment may be effective. To the best of our knowledge, however, no research has been done yet to investigate the effects of such a combined in- and outpatient treatment program, but the development of such a program seems opportune.

Conclusion

In conclusion, our data suggest that there are useful psychological treatments for PNES patients and that they may profit from a comprehensive, multidisciplinary, inpatient treatment program following cognitive behavioural principles. Our treatment resulted not only in a decrease of seizures, but also in a decrease of general psychopathology, anxiety, depression and dissociation, and an increase in coping abilities. This applies for seizure-free patients more strongly than for patients with ongoing seizures after treatment, implying that seizure cessation should be an important focus of treatment.

Acknowledgments

We thank Erna Haverkort and Paul Endert for data gathering and Ann Tierlier-Long for manuscript preparation.

References

1. Andermann F. Non-epileptic paroxysmal neurologic events. In: Gates JR, Rowan AJ, editors. *Non-epileptic seizures*. 2nd ed. Boston: Butterworth-Heinemann; 2000. p. 51—69.
2. LaFrance WC, Benbadis SR. Avoiding the costs of unrecognized psychological nonepileptical seizures. *Neurology* 2006;**66**: 1620—1.
3. Martin RC, Gilliam FG, Kilgore M, Faught E, Kuzniecky R. Improved care resource utilization following video-EEG-confirmed diagnoses of nonepileptic psychogenic seizures. *Seizure* 1998;**7**:385—90.
4. Benbadis SR, Hauser WA. An estimate of the prevalence of psychogenic non-epileptic seizures. *Seizure* 2000;**9**: 280—1.
5. Gumnit RJ. Psychogenic seizures. In: Wyllie E, editor. *The treatment of epilepsy: principles and practice*. Philadelphia: Lea & Febiger; 1993. p. 692—6.
6. Reuber M, Fernandez G, Bauer J, Helmstaedter C, Elger CE. Diagnostic delay in psychogenic nonepileptic seizures. *Neurology* 2002;**58**:493—5.
7. LaFrance WC, Barry JJ. Update on treatments of psychological nonepileptic seizures. *Epilepsy Behav* 2005;**7**:364—74.
8. Bowman ES, Markand ON. The contribution of life events to pseudoseizure occurrence in adults. *Bull Menninger Clin* 1999;**63**:70—88.
9. Rush MD, Morris GL, Allen L, Lathrop L. Psychological treatment of nonepileptic events. *Epilepsy Behav* 2001; **2**:277—83.
10. Goldstein LH, Deale AC, Mitchell-O'Malley SJ, Toone BK, Mellers JDC. An evaluation of cognitive behavioural therapy as a treatment for dissociative seizures. *Cogn Behav Neurol* 2004;**17**:41—9.
11. Zaroff CM, Myers L, Barr WB, Luciano D, Devinsky O. Group psychoeducation as treatment for psychological nonepileptic seizures. *Epilepsy Behav* 2004;**5**:587—92.
12. Aboukasm A, Mahr G, Gahry BR, Thomas A, Barkley GL. Retrospective analysis of the effects of psychotherapeutic interventions on outcome of psychogenic nonepileptic seizures. *Epilepsia* 1998;**39**:470—3.
13. Chemali Z, Meadows M. The use of eye movement desensitization and reprocessing in the treatment of psychogenic seizures. *Epilepsy Behav* 2004;**5**:784—7.
14. McDade G, Brown SW. Non-epileptic seizures: management and predictive factors of outcome. *Seizure* 1992;**1**:7—10.
15. LaFrance WC, Devinsky O. The treatment of nonepileptic seizures: Historical perspectives and future directions. *Epilepsia* 2004;**45**(S2):15—21.
16. Baker GA, Brooks JL, Goodfellow L, Bodde N, Aldenkamp A. Treatments for non-epileptic attack disorder (Review). *Cochrane Database Syst Rev*)2007;(1). 10.1002/14651858.CD006370. [Art. No.: CD006370].
17. Walczak TS, Papacostas S, Williams DT, Scheuer ML, Lebowitz N, Notarfrancesco A. Outcome after diagnosis of psychogenic nonepileptic seizures. *Epilepsia* 1995;**36**:1131—7.
18. Guberman A. Psychogenic pseudoseizures in non-epileptic patients. *Can J Psychiatry* 1982;**27**:401—4.

19. Kim CM, Barry JJ, Zeifert PA. The use of inpatient medical psychiatric treatment for nonepileptic events. *Epilepsia* 1998;**39**(Suppl. 6):242—3.
20. Quigg M, Armstrong RF, Farace E, Fountain NB. Quality of life outcome is associated with cessation rather than reduction of psychogenic nonepileptic seizures. *Epilepsy Behav* 2002;**3**: 455—9.
21. Reuber M, Mitchell AJ, Howlett S, Elger CE. Measuring outcome in psychogenic nonepileptic seizures: how relevant is seizure remission? *Epilepsia* 2005;**46**:1788—95.
22. Kuyk J, Swinkels WAM, Spinhoven P. Psychopathology in patients with nonepileptic seizures with and without comorbid epilepsy: how different are they? *Epilepsy Behav* 2003;**4**:13—8.
23. Derogatis LR. *Administration, scoring and procedures manual for the R(evised) version*. Baltimore: John Hopkins University School of Medicine, Clinical Psychometrics Research Unit; 1977
24. Arrindell WA, Ettema JHM. *Handleiding SCL-90*. Lisse: Swets & Zeitlinger; 1986.
25. Beck AT, Rush AJ, Shaw BF, Emery G. *Cognitive therapy of depression*. New York: Wiley & Sons; 1979.
26. Bouwman TK, Luteijn F, Albersnagel FA, Van der Ploeg FAE. Enige ervaringen met de Beck Depression Inventory (BDI). *Gedrag -Tijdschrift voor Psychologie* 1985;**13**:13—24.
27. Spielberger CD, Gorsuch RL, Lushene RE. *Manual for the State-Trait Anxiety Inventory (self-evaluation questionnaire)*. Palo Alto, CA: Consulting Psychologists Press; 1970 .
28. van der Ploeg HM, Defares PB, Spielberger CD. *Handleiding bij de Zelf Beoordelings Vragenlijst, ZBV*. Lisse: Swets & Zeitlinger; 1988.
29. Schreurs PJG, van de Willige G, Brosschot JF, Tellegen B, Graus GMH. *Herziene Handleiding UCL*. Lisse: Swets & Zeitlinger; 1993.
30. Ware JE, Sherbourn CD. The MOS 36-item short-form health survey (SF-36): Conceptual framework and item selection. *Med Care* 1992;**30**:473—83.
31. Aaronson NK, et al. Translation, validation and norming of the Dutch language version of the SF-36 health survey in community and chronic disease populations. *J Clin Epidemiol* 1998;**51**:1055—68.
32. Vanderlinden J, Van Dyck R, Vandereycken W, Vertommen H, Verkes RJ. The dissociation questionnaire: development and characteristics of a new self-reporting questionnaire. *Clin Psychol Psychother* 1993;**1**:21—7.
33. Shen W, Bowman ES, Markand ON. Presenting the diagnosis of pseudoseizures. *Neurology* 1990;**40**:756—9.
34. Rifkin A. Randomized controlled trials and psychotherapy research. *Am J Psychiatry* 2007;**164**:7—8.
35. Lesser RP. Treatment and outcome of psychogenic nonepileptic seizures. *Epilepsy Curr* 2003;**3**:198—200.